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Title: Changes in Lactate Kinetics Underpin Soccer Performance Adaptations to Cycling-Based Sprint Interval Training

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Abstract

In adolescent soccer, 23% of the distance covers happens at speeds above onset of blood lactate accumulation which suggests that lactate kinetics may be important for soccer performance. We sought to determine the effectiveness of sprint interval training (SIT) on changing performance and lactate kinetics in adolescent soccer players. Thirteen elite soccer academy players (age 15 ± 0.5y) underwent baseline testing (0-10m and 10-20m sprint performance, Wingate anaerobic Test (WaNT) with blood lactate measurements and incremental VO2 peak test) before being allocated to control or SIT group. The control group maintained training whilst the HIT group carried out twice-weekly all-out effort cycle sprints consisting of 6 x 10sec sprint with 80sec recovery. There were significant time x group interactions for 10- 20m sprint time (Control pre: 1.32 ± 0.07s post: 1.35 ± 0.08s; SIT pre: 1.29 ± 0.04s post: 1.25 ± 0.04s; p=0.01), Peak Power (Control pre: 13.1 ± 1.3W.kg^{-1} post: 13.2 ± 1.47 W.kg^{-1}; SIT pre: 12.4 ± 1.3 W.kg^{-1} post: 15.3 ± 0.7W.kg^{-1}; p=0.01) and time to exhaustion (Control pre: 596 ± 62s post: 562 ± 85s; SIT pre: 655 ± 54s post: 688 ± 55s; p=0.001). The changes in performance were significantly correlated to changes in lactate kinetics (power: r=0.55; 10-20m speed: r=-0.54; time to exhaustion: r=0.55). Therefore, cycle based SIT is an effective training paradigm for elite adolescent soccer players and the improvements in performance are associated with changes in lactate kinetics.

Keywords: Metabolism, Team Sport, Youth, Physiology

Introduction

Soccer is an intermittent sport with bouts of high intensity activity followed by longer periods of low to moderate intensity exercise (Aslan et al., 2012). Average heart rate during game play in 17-year-old players is approximately 85% of maximum with recorded blood lactate concentrations between 4 and 8 $mmol \cdot l^{-1}$ (Aslan et al., 2012). This shows a reliance on anaerobic glycolysis during periods of high intensity activity. In fact, Aslan et al., 2012 have demonstrated that 23% of the distance travelled during a game occurs at running speeds above the onset of lactate accumulation (set at 4 $mmol \cdot l^{-1}$). Therefore, a player's ability to metabolise lactate could be a key determinant of soccer performance, allowing a greater game play intensity with lower lactate accumulation (Best, Simon, Niess, & Striegel, 2013). Indeed, blood lactate concentration is greater in elite soccer players compared to non-elite players due to the higher number of high intensity activities at the elite level (Mohr, Krustrup, & Bangsbo, 2003). Despite the importance of lactate for high intensity activities, there is a drop in blood lactate concentration between the first and second half of game play that coincides with a drop in the number of sprinting and high intensity efforts (Aslan et al., 2012). It has been suggested that the distance a player covers at high intensity may reflect their ability to maintain lactate balance as assessed by lactate threshold (Edwards, Clark, & Macfadyen, 2003). Therefore, targeting lactate metabolism through training may be beneficial for soccer performance.

In young soccer players (13.5 ± 0.4 years), a 5-week running-based high-intensity interval training programme (HIIT) has been shown to significantly increase (7%) maximal oxygen uptake ($\dot{V}O_2max$) and reduce (4.2%) 1000-m running time compared to non-significant changes (+1.9% and -2.0%, respectively) in the high-volume endurance training (HVT) group (Sperlich et al., 2011). Despite the reduced weekly training time of 1.5-2 hours, the HIIT group also significantly improved sprint time over 20-, 30- and 40-m distances with no changes observed in jumping performance (Sperlich et al., 2011). Similarly, a study by (Faude, Schnittker, Schulte-Zurhausen, Müller, & Meyer, 2013) compared the training effects between HIIT and HVT programmes on an intra-individual basis in high-level youth soccer players (15.9 ± 0.8 years). In agreement with (Sperlich et al., 2011), (Faude et al., 2013)found that even with a 70% reduction in the total training time HIIT seems to be as effective

as the HVT at improving endurance capacity with significant decreases in jump heights reported after both interventions. A recent meta-analysis involving 232 young soccer players (16.2 ± 1.6 years) has found that HIIT and small-sided games (SSG) have equally beneficial effects on variables related to the endurance and soccer-specific performance, but no impact on sprint and jumping performance, or repeated sprint ability (Kunz, Engel, Holmberg, & Sperlich, 2019). These findings suggest that HIIT offers a time-efficient training modality to improve young soccer players' performance, but exact physiological mechanisms behind its effectiveness are still not fully understood.

The inclusion of generic running-based training over a 5-week preseason period has been shown to produce a rightward shift in the blood lactate curve, with a greater speed associated with the onset of blood lactate accumulation in elite youth soccer players (15.0 ± 0.5 years) (Best et al., 2013). This is similar to the adaptations shown in adult triathletes in response to 2 weeks of cycling-based sprint interval training (SIT) (Jakeman, Adamson, & Babraj, 2012). However, an increase in acute high-speed running distances have been shown to significantly increase non-contact injury risk in elite youth soccer players (Bowen, Gross, Gimpel, & Li, 2017). Therefore, a cycling-based SIT may be a more desirable training approach to improve lactate kinetics and performance in soccer players. Given that the lactate kinetic response is similar between adolescents and adults post Wingate Anaerobic Test (WAnT) (Ralph Beneke, Hütler, Jung, & Leithäuser, 2005) then a similar adaptation to lactate metabolism and kinetics may be expected following cycling-based SIT in adolescent athletes. However, no previous studies have studied lactate kinetics in adolescent soccer players in response to cycling-based SIT.

The aim of the current study was to determine the effects of a six week, twice-weekly cycling-based SIT protocol (twelve sessions in total) on lactate kinetics and performance characteristics in adolescent male soccer players. It was hypothesised that cycling-based SIT would lead to increased rate of lactate clearance and improve fitness components related to soccer performance.

Methods

Study Design

A two-group pre-post-test research design was employed in the study. To assess responses to cycling-based SIT, the participants were tested pre- and post-training with testing being performed over a seven day period and within 72 hours of completing training. At baseline the participants completed a 20-m sprint test, Wingate test with pre- and post-exercise blood lactate measurements and an incremental $\dot{V}O_2$peak cycling test. Post-training tests were performed in the same order as baseline testing. For a given participant, each training and testing protocol was performed within 2 hours of the same time of the day. The participants were also asked to refrain from vigorous training for 24 hours before each test and to maintain their normal training and diet routine throughout the study period.

Participants

Sixteen elite male under 17 (U17) players, as defined by being academy players at a professional club with 4 years' experience within the academy structure, were recruited for this study (age: 15 ± 0.5 years; $\dot{V}O_2$peak: 56.1 ± 5.6 ml· kg^{-1}·min^{-1}, Table 1) and were randomly allocated to either the training or the control group. One participant dropped out of the training group due to an injury sustained during a game and two participants dropped out of the control group after being released by the club, leaving 7 in the training group and 6 in the control group (Table 1). The control group were asked to maintain their normal activities whilst the training group took part in twice weekly cycling-based SIT, as well as continuing their normal training. All participants and parents/guardians were informed of the study verbally and in writing. Parents/guardians' written informed consent and participants' assent were obtained prior to the study. The study had ethical approval from Abertay University Ethics Committee and conducted in accordance with the Declaration of Helsinki (World Medical Association, 2013).

Baseline Testing

On the initial visit, participants reported to the laboratory at a time suitable for them after a 4-h fast. Height was measured (SECA 217 stadiometer, United Kingdom) to the nearest 0.1 cm prior to stepping onto a calibrated bioelectrical impedance meter (SC-330ST Tanita Body Composition Analyser, Tanita Europe BV, Amsterdam, Netherlands) where body fat mass and lean body mass were recorded to the nearest 0.1 kg (Table 1).

Twenty metre sprint test

The 20-m sprint was carried out indoors on a 4G football surface. Single light beam timing gates (Brower Speed Trap II; Brower, Utah, USA) were set up at 0-, 10- and 20-m, with the gates set at a height of 140 cm, and the participants began from a standing start 2-m behind the zero light gate and sprinted as fast as possible through the three light gates. Single light gates can give false data by the arm or leg cutting the light prior to the body. However, when the gates are set at belt height then this minimises the risk of leg or arm breaks and produces reliable timings (Altmann et al., 2017). Time for 0-10 m and 10-20 m were recorded and 1 minute recovery was given prior to repeating the sprint. The average value of three attempts is reported.

Wingate Anaerobic test (WAnT)

The test was carried out on a cycle ergometer (Monark Ergomedic 891E Bike Ergometer, Vansbro, Sweden) designed for immediate load resistance with foot slippage prevented through the use of toe clips. The ergometer seat height was adjusted to allow for a full leg extension at the bottom of the cycle stroke and participants then performed a 3-minute warm-up cycling at 60 W. Participants were given a "3-2-1-Go" countdown and instructed to sprint "all-out" for 30 sec. The 30-s sprint started once the participants reached 100 $r \cdot min^{-1}$, when resistance of 7.5% of body mass was automatically released by the cycle ergometer. Verbal encouragement was supplied to participants throughout the 30-s maximal intensity sprint (Zupan et al., 2009). No cool down was given after the test to allow for measurement of lactate kinetics. Peak power (PP) and average power (AP) during each 30-s sprint was automatically calculated using software (Monark Anaerobic Test Software v.2.24.2, Monark Exercise AB).

Blood lactate concentration was determined via fingertip blood samples (Lactate pro, Arkray Inc., Kyoto, Japan). Blood lactate concentration was measured prior to the WAnT and then 0, 1, 3, 5, 10, 15, 20, 25 and 30 min after the WAnT. The skin was punctured using an Accu-check single use lancet (Roche Diagnostics, UK) and pressure applied to the finger to draw the blood. The initial drop was discarded and the second drop was taken for analysis. Blood lactate kinetics were modelled using the

four parameter model that has been previously used in adolescents (Beneke et al., 2005) and fitting the following bi-exponential function:

$$BLC(t) = A \cdot k_1/k_2 - k_1 \cdot (e^{-k1 \cdot t} - e^{-k2 \cdot t}) + BLC_0 \quad (1)$$

Where A is extravascular release of lactate from exercise metabolism, k_1 is the rate of lactate accumulation, k_2 is the rate of lactate clearance, t is time and BLC_0 is blood lactate concentration prior to the WAnT. Maximum blood lactate concentration (BLC_{max}), time to maximum blood lactate concentration ($TBLC_{max}$) and the turn point (TP) were blood lactate concentration begins to decrease were described by mono-exponential functions:

$$BLC_{max} = BLC_0 + A \cdot (k_1/k_2)^{k2/k1-k2} \quad (2)$$

$$TBLC_{max} = 1/k_1-k_2 \cdot \ln(k_1/k_2) \quad (3)$$

$$TP = 2/k_1-k_2 \cdot \ln(k_1/k_2) \quad (4)$$

$\dot{V}O_2$peak cycling test

Participants performed an incremental test to exhaustion (TTE) to determine their $\dot{V}O_2$peak on a cycle ergometer (Monark Ergomedic 894E, Varberg, Sweden). Prior to starting the test, participants were connected to a breath by breath oxygen analyser (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany). The test started at an initial power output of 70W, with an additional 35W increase every minute until volitional exhaustion or the participants could not maintain 70 r·min^{-1} despite strong verbal encouragement. Exercise duration at exhaustion was recorded to the nearest second and was defined as time to exhaustion (TTE). $\dot{V}O_2$peak was taken as the highest 30 sec average over the incremental test.

Sprint Interval Training

Seat height was adjusted to allow a full leg extension at the bottom of the cycle stroke and participants cycled for 1 minute at 60W. The participants then performed 6 x 10 sec "all-out" sprints against a resistance of 7.5% of body mass, with 80 sec recovery between each sprint (1:8 work-to-rest ratio). The sprint started once the participant reached 120 r·min^{-1} and the resistance was automatically

dropped by the cycle ergometer (Monark Ergomedic 894E, Varberg, Sweden). Participants then cycled for 30 sec against no resistance prior to getting off the bike. Short duration sprints have been shown to be as effective as longer duration sprints at eliciting performance benefits (Yamagishi & Babraj, 2017) and 10 sec sprints have been shown to be effective in long distance runners (Kavaliauskas, Aspe, & Babraj, 2015). Training sessions were carried out twice per week over 6 weeks and each session lasted a total of 9 minutes (12 sessions in total).

Post-Testing

All tests were repeated within 2 hours of the same time of the day, with the same fast period and in the same order as baseline after 6 weeks. There was a minimum of 72 h between the last training session and retesting.

Data Analysis

All data are presented as mean ± SD. The bi-exponential model was fitted using QtiPlot to generate values for A, k_1 and k_2. All statistical analysis was carried out using IBM SPSS Statistics for Windows, version 23.0 (IBM Corp., Armonk, N.Y., USA). Before conducting parametric tests, a Shapiro-Wilk test was performed to ensure that all values were normally distributed. An independent samples *t*-test was run to determine differences at baseline between the groups. There was no significant difference at baseline between the groups. A repeated measures ANOVA was then performed to determine time x group interaction. When there was a significant time x group interaction an ANCOVA was run with pre values as covariates to determine the difference between groups. Pearsons correlations were used to assess the relationships between lactate kinetic parameters and performance measures. Significance was accepted as $p<0.05$. Partial eta squared ($\eta 2$) was defined as 0.02 small, 0.13 medium and 0.26 large effect sizes as proposed by Bakeman (2005).

Results

Sprint performance – There was no significant difference between groups at baseline for 0-10 m ($p = 0.37$ control vs. SIT at baseline; Figure1A) and 10-20 m ($p = 0.45$ control vs. SIT at baseline; Figure 1B) sprint times. Sprint performance remained statistically unchanged in the control group (Figure 1A

or B). Following 6 weeks of cycling-based SIT, there was no significant time x group interaction ($p = 0.48$) and no significant change in 0-10 m sprint performance in the SIT group compared to control, although there was a medium effect size ($p = 0.17$; $\eta 2 = 0.18$; Figure 1A). There was a significant time x group interaction ($p = 0.004$), with a significant improvement in 10-20 m sprint performance in the SIT group compared to control, with a large effect size ($p = 0.01$; $\eta 2 = 0.56$; Figure 1B).

Endurance performance – There was no significant difference between groups at baseline for $\dot{V}O_2$peak ($p = 0.21$ control vs. SIT at baseline; Figure 2A) and TTE ($p = 0.69$ control vs. SIT at baseline; Figure 2B). Endurance performance remained statistically unchanged in the control group (Figure 2A and B). Following 6 weeks of cycling-based SIT, there was a significant time x group interaction ($p = 0.049$), with a trend for improvement in $\dot{V}O_2$peak in the SIT group compared to control, with a large effect size ($p = 0.08$; $\eta 2 = 0.27$; Figure 2A). There was a significant time x group interaction ($p = 0.001$) with a significant improvement in TTE in the SIT group compared to control, with a large effect size ($p = 0.001$; $\eta 2 = 0.59$; Figure 2B).

WAnT performance – There was no significant difference between groups for PP (Table 1) and AP at baseline (Table 1). WAnT performance remained statistically unchanged in the control group (Table 1). Following 6 weeks of cycling-based SIT, there was a significant time x group interaction in both PP ($p = 0.02$) and AP ($p = 0.04$), with a significant improvement in the SIT group compared to control for PP and AP, with a large effect size for both (Table 1).

Lactate kinetics – There was no significant difference between groups for A, k_1, k_2, BLC_{max}, $TBLC_{max}$ and TP at baseline (Table 1). No significant changes occurred in any of the lactate kinetic parameters in the control group (Table 1). Following 6 weeks of cycling-based SIT, there was a significant time x group interaction for A ($p = 0.049$) and k_2 ($p = 0.001$), with a significant change in the SIT group post-training compared to control (Table 1). There were no other significant differences for the other kinetic parameters (k_1 $p = 0.82$; BLC_{max} $p = 0.26$; $TBLC_{max}$ $p = 0.12$; TP $p= 0.12$) although there was a large effect size for BLC_{max}, $TBLC_{max}$ and TP (Table 1).

Correlation – There were significant positive correlations between both A and BLC_{max} with peak and average power during the WAnT (Table 2), and significant negative correlations between A and

BLC_{max} with 0-10 m and 10-20 m sprint time (Table 2). There were significant positive correlations between k_2, $TBLC_{max}$ and TP with $\dot{V}O_2$peak and TTE (Table 2). There was no correlation between k_1 and any performance variable (Table 2).

Discussion

The purpose of this study was to determine the effects of a six week, twice-weekly cycling-based SIT intervention on lactate kinetics and performance characteristics in competitive male adolescent soccer players. The main findings were that this type of SIT significantly improved performance in 10-20 m sprint time (-4%), PP (+24%) and AP (+5%) during WAnT, and TTE (+5%). Furthermore, improvements in different aspects of performance were correlated with changes in lactate kinetics.

Recently, HIIT has found to be a time-efficient alternative to longer training protocols, such as SSG and low- or moderate-intensity continuous exercise in young athletes (15.5 ± 2.2 years) (Engel, Ackermann, Chtourou, & Sperlich, 2018). Cycling-based SIT has the advantage of reducing joint impact during training and reducing total time commitment to fitness training even further, which will free time for technical and tactical training for adolescent players. The training duration in the current study was shorter than those reported in previous running-based HIIT studies involving adolescent soccer players (18 minutes vs. 29-33 minutes used by Sperlich et al., (2011); Faude et al., (2013)). Therefore, it appears that doing cycling-based SIT training twice-weekly (18 minutes) in addition to the regular soccer training sessions can help players develop aerobic and anaerobic capacity.

Sprint and Power Performance

Soccer is an intermittent game, which requires the athlete to generate power for sprinting and other explosive movements (Cometti, Maffiuletti, Pousson, Chatard, & Maffulli, 2001). Following 6 weeks of cycling-based SIT, there was a significant decrease (-4%) in 10-20 m sprint time but no change in 0-10 m sprint time (Figure 1A&B). Performance in 0-10 m and 10-20 m sprint times are similar to those reported for other elite adolescent soccer players (Tomáš, František, Lucia, & Jaroslav, 2014). In contrast to the current study, a 7-week intervention using repeated-sprint training at 90% or 100% of maximal sprint velocity did not improve single-sprint and repeated-sprint performance in

adolescent soccer players (Haugen et al., 2015). Speed and explosive power are crucial for the development of adolescent soccer players (Reilly, Bangsbo, & Franks, 2000) and it has been suggested that sprint performance is resistant to training adaptations (Haugen et al., 2015). From the present study it would seem that intensity (i.e., 'all-out' effort) is a key determinant for sprint adaptation to training. However, more studies are required to determine why significant improvement was only observed between 10-20 m with no change in 0-10 m sprint time.

Following 6 weeks of cycling-based SIT, there was a significant increase in both peak power (PP) and average power (AP) during the WAnT (Table 1). PP and AP values in this study are similar to the results recorded in competitive U17 soccer players (Nikolaïdis, 2011). Significant improvements in PP and AP after 12 sessions of 4-6 15-s SIT have been previously reported in young healthy males (Zelt et al., 2014). It has been suggested that PP during WAnT is a measure determined predominantly by phosphocreatine (PCr) degradation and AP predominantly reflects anaerobic glycolysis (Nikolaïdis, 2011). However, this is an oversimplification of the energetics and even during the first 5-s of a sprint; glycolysis is a key component of energy production (Beneke, Pollmann, Bleif, Leithäuser, & Hütler, 2002). This suggests that improvements in both PP and AP are related to an increase in the energy production from anaerobic glycolysis. For example, following a 7-week repeated 5-s sprint cycling training programme, there was a greater activity of phosphofructokinase and lactate dehydrogenase in skeletal muscle in young moderately active adults (Linossier et al., 1997). Alternatively, changes in neuromuscular activation may account for changes in PP, with 2 weeks of sprint cycling resulting in greater neuromuscular activation (Martinez-Valdes, Falla, Negro, Mayer, & Farina, 2017). No studies have been carried out looking at skeletal muscle adaptation to SIT specifically in children or adolescents. However, given that glycolytic enzyme activity in adolescents is similar to young adults (Berg, Kim, & Keul, 1986) and the improvements in performance recorded are similar to those seen in adults then it seems reasonable to assume similar skeletal muscle adaptation in adolescent soccer players.

Endurance Performance

Aerobic power has been identified as a key component of elite soccer player development (Ostojic, 2004), with improvements in $\dot{V}O_2$max associated with greater distance covered during game play (Helgerud, Engen, Wisløff, & Hoff, 2001) and at a greater running speed (E. Rampinini et al., 2007) Following 6 weeks of cycling-based SIT, $\dot{V}O_2$peak increased by 9% with a significant increase in TTE (Figure 2A&B). At baseline the reported $\dot{V}O_2$peak values are similar to other values recorded for elite adolescent soccer players (Helgerud et al., 2001). The magnitude of change (+9%) in $\dot{V}O_2$peak is greater than the 5% improvement reported in recreationally active adolescents (15.1 ± 0.3 years) after six 30-s cycle sprint intervals (Barker, Day, Smith, Bond, & Williams, 2014) or that reported in prepubertal soccer players following strength and high-intensity training (Ferrete, Requena, Suarez-Arrones, & De Villarreal, 2014). However, the size of change is similar to that reported rate in elite junior soccer players after 16 sessions of a 4 x 4 min running protocol at 90-95% of maximal heart (Helgerud et al., 2001). The increase seen in the present study occurs with a much lower training volume (120 sec sprinting per week) compared to that used by (Helgerud et al., 2001) consisting of 32 minutes sprinting per week. Indeed, a recent study by Yamagishi & Babraj (2017) has shown that sprint duration during cycling-based SIT does not affect endurance adaptation in adults. Improvements in $\dot{V}O_2$peak following short duration SIT in young recreationally active adults have been demonstrated to be peripheral and reflect greater mitochondrial density and activity post-training (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005). Given that sprint duration does not appear to affect training adaptations, this suggests that the mitochondrial adaptation must be driven by similar events in the skeletal muscle during training. However, more research is needed to determine the mechanisms of adaptation.

Fatigue is associated with a decline in soccer performance, and players with a lower Yo-Yo intermittent recovery test distance have a greater decline in soccer performance during game play (Rampinini et al., 2008). TTE during the incremental test reflects peripheral fatigue with loss of EMG amplitude towards volitional exhaustion (Shinohara, Kouzaki, Yoshihisa, & Fukunaga, 1997). Therefore, significant improvements (+ 5%) in TTE in this study (Figure 2B) reflect a better ability to resist peripheral fatigue in adolescent soccer players. This is likely to represent changes in

mitochondrial function with faster on oxygen kinetics following SIT (Bailey, Wilkerson, Dimenna, & Jones, 2009), which may delay the onset of depletion of muscle high-energy phosphates and accumulation of fatigue related metabolites (Vanhatalo, Fulford, Dimenna, & Jones, 2010).

Lactate Kinetics

Following 6 weeks of cycling-based SIT in adolescent soccer players, we report a significant change in lactate kinetics (Table 1). Specifically, there was a significant increase in extravascular release of lactate (A), maximum blood lactate concentration (BLC_{max}) and an increase in the rate of lactate disappearance (k_2) but no change in rate of lactate appearance (k_1). There were also significant reductions in the time to blood lactate peak ($TBLC_{max}$) and blood lactate turning point (TP) following training. To date, no other studies have looked at lactate kinetics in adolescent athletes in response to SIT. However, these findings are similar to those reported in adults following 6 weeks of 2-minute running intervals at an intensity of 90-100% of maximal aerobic speed (MAS) (Gharbi et al., 2008). This suggests that duration of interval is not a key training variable for whole body lactate kinetics. Changes in lactate clearance have been associated with increased MCT1 content in skeletal muscle (Thomas, 2004) and this has been shown to be increased following high intensity exercise (Pilegaard et al., 1999).

In the current study, there are significant negative correlations between A and BLC_{max} and overall sprint time for 0-10 m and 10-20 m (Table 2). The greater the value for A and BLC_{max}, the faster the sprint time, which suggests that sprint speed is associated with the ability of skeletal muscles to generate lactate from anaerobic glycolysis. In adults, improvement in sprint performance has been shown to be accompanied by an increase in the post-exercise muscle lactate concentration (Nevill, Boobis, Brooks, & Williams, 1989) and running sprint time has been moderately correlated to post-exercise peak lactate concentration in highly trained youth football players (Mujika, Spencer, Santisteban, Goiriena, & Bishop, 2009). There were significant positive correlations between A and BLC_{max} and WAnT PP and AP (Table 2). This suggests that the ability to generate adenosine triphosphate (ATP) from anaerobic glycolysis is an important determinant of power production during a WAnT. Indeed, a relationship between post-WAnT lactate concentration and AP has been shown in

adolescents (Falgairette, Bedu, Fellmann, Van-Praagh, & Coudert, 1991). Similarly, the energetics of the WAnT in young adults demonstrate that approximately 50% of the total energy turnover comes from anaerobic glycolysis and the proportion of anaerobic glycolysis utilised explains >80% of the variation in PP and AP production (Beneke et al., 2002). In contrast, endurance adaptations ($\dot{V}O_2$peak and TTE) were significantly correlated to k_2, $TBLC_{max}$ and TP. This suggests that endurance performance is related to changes in the ability to process lactate for energy production. In young adults, MAS and TTE are shown to be strongly associated with the rate of lactate removal (Gharbi et al., 2008). This improvement in lactate metabolism has been shown to result in a rightward shift in the blood lactate curve during incremental exercise and is associated with greater capillarisation of the skeletal muscle (Messonnier et al., 2002). The improvements in performance are linked to changes in lactate dynamics, therefore it seems a necessity that soccer specific training should seek to enhance lactate kinetics to improve player performance. Failure to structure training to enhance lactate metabolism may lead to increased risk of injury due to fatigue during the later stages of a game due to poor kinetic chain control (Lehnert et al., 2017). These adaptations are key for adolescent player development and the low impact nature of cycling minimises the risk of overuse injuries (Gist, Fedewa, Dishman, & Cureton, 2014).

In conclusion, the present study demonstrates that cycling-based SIT is an effective way to produce rapid physiological adaptations that are desirable for soccer performance by adding just 18 minutes per week to the regular soccer training sessions. The adaptations of speed and power would appear to be determined by the ability of the body to activate anaerobic glycolysis; in contrast, endurance adaptations are related to the extent of lactate utilisation. From the data in the current study, training should seek to promote changes in lactate kinetics and failure to do so may impede an adolescent athlete from progressing.

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Table 1. Participant characteristics, WAnT performance and lactate kinetics post-WAnT (PP=peak power; AP= average power; A=extravascular release of lactate from exercise; k_1=rate of lactate accumulation; k_2=rate of lactate clearance; BLC_{max}=maximum blood lactate concentration; $TBLC_{max}$=time to maximum blood lactate accumulation; TP= turn point; η 2 = partial eta squared). a: $p<0.05$ group x time interaction; b: $p<0.01$ group x time interaction; c: $p<0.02$ ANCOVA post-SIT compared to post-control

	Control (pre)	Control (post)	Cycle SIT (pre)	Cycle SIT (post)	η^2
Age (y)	15.0 ± 0.6	15.0 ± 0.6	15.0 ± 0.5	15.0 ± 0.5	*0.00*
Height (cm)	175.0 ± 5.1	175.2 ± 4.9	179.9 ± 7.7	180.0 ± 7.6	*0.00*
Mass (kg)	54.7 ± 4.5	56.2 ± 4.6	60.6 ± 4.1	62.6 ± 4.8	*0.00*
Fat Free Mass (kg)	11.9 ± 2.0	10.8 ± 1.0	14.0 ± 2.3	12.3 ± 1.7	*0.02*
PP ($W \cdot kg^{-1}$)	13.1 ± 1.3	13.2 ± 1.5	12.4 ± 1.6	15.3 ± 0.7 [a,c]	*0.48*
AP ($W \cdot kg^{-1}$)	8.8 ± 0.3	8.6 ± 0.5	9.0 ± 0.4	9.5 ± 0.3 [a,c]	*0.45*
A ($mmol \cdot l^{-1}$)	15.8 ± 2.2	13.9 ± 3.2	15.4 ± 3.8	18.7 ± 3.1 [a,c]	*0.41*
K_1 (min^{-1})	0.71 ± 0.36	0.70 ± 0.28	0.81 ± 0.33	0.86 ± 0.22	*0.10*
K_2 (min^{-1})	0.10 ± 0.03	0.08 ± 0.03	0.08 ± 0.03	0.12 ± 0.04 [a,c]	*0.68*
BLC_{max} ($mmol \cdot l^{-1}$)	11.9 ± 0.8	11.8 ± 3.1	13.2 ± 3.4	15.4 ± 1.7	*0.39*
$TBLC_{max}$ (min)	3.5 ± 1.3	3.8 ± 1.2	3.5 ± 1.1	2.9 ± 0.5	*0.34*
TP (min)	7.0 ± 2.5	7.6 ± 2.3	7.0 ± 2.1	5.6 ± 1.1	*0.34*

Table 2. Correlation between training outcomes and lactate kinetic parameters. PP=peak power; AP= average power; A=extravascular release of lactate from exercise; k_1=rate of lactate accumulation; k_2=rate of lactate clearance; BLC_{max}=maximum blood lactate concentration; $TBLC_{max}$=time to maximum blood lactate accumulation; TP= turn point; TTE= incremental time-to-exhaustion. a: $p<0.05$ main effect, training variable to lactate kinetic response; b: $p<0.01$ main effect, training variable to lactate kinetic response

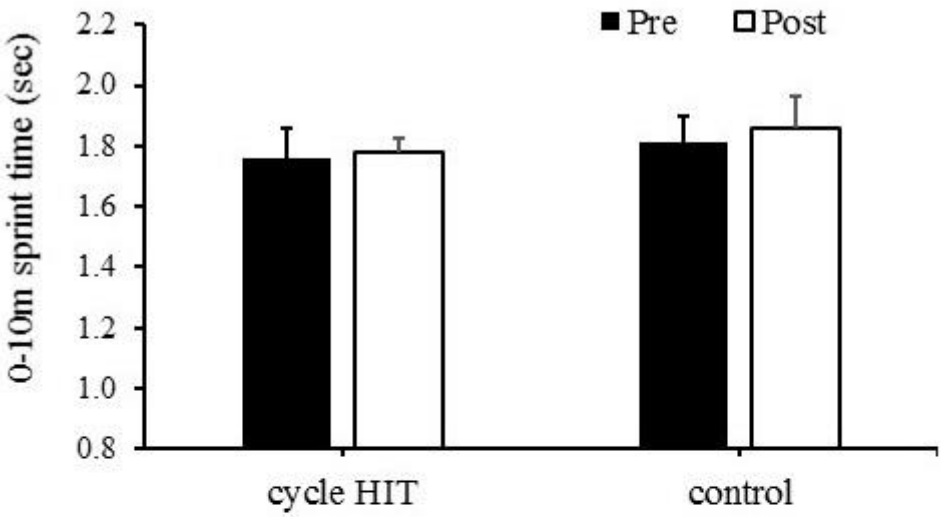
Table 2: Correlation between training outcome and lactate kinetic parameter. PP=peak power; AP= average power; A=extravascular release of lactate from exercise; k_1=rate of lactate accumulation; k_2=rate of lactate clearance; BLC_{max}=maximum blood lactate concentration; $TBLC_{max}$=time to maximum blood lactate accumulation; TP= turn point; TTE= incremental time to exhaustion ([a]$p \leq 0.05$ training variable to lactate kinetic response; [b]$p \leq 0.01$ training variable to lactate kinetic response)

	VO_2 peak	TTE	Peak Power	Average Power	0-10m sprint time	10-20m sprint time
A	-0.01	0.28	0.55[b]	0.59[b]	-0.52[b]	-0.54[b]
k_1	0.24	0.13	0.03	-0.16	0.03	0.01
k_2	0.45[a]	0.55[b]	0.13	0.24	0.04	-0.25
$BLAC_{max}$	-0.07	0.17	0.58[b]	0.54[b]	-0.50[b]	-0.45[b]
$TBLAC_{max}$	-0.45[a]	-0.39[a]	0.12	0.01	-0.05	0.08
TP	-0.45[a]	-0.39[a]	0.12	0.01	-0.05	0.08

Figure 1. Change in sprint performance; A: 0-10 m sprint time; B: 10-20 m sprint time. **p<0.001 ANCOVA post-SIT compared to post-control

Figure 2. Change in endurance performance; A: $\dot{V}O_2$peak; B: time to exhaustion. **p<0.001 ANCOVA post-SIT compared to post-control

1A



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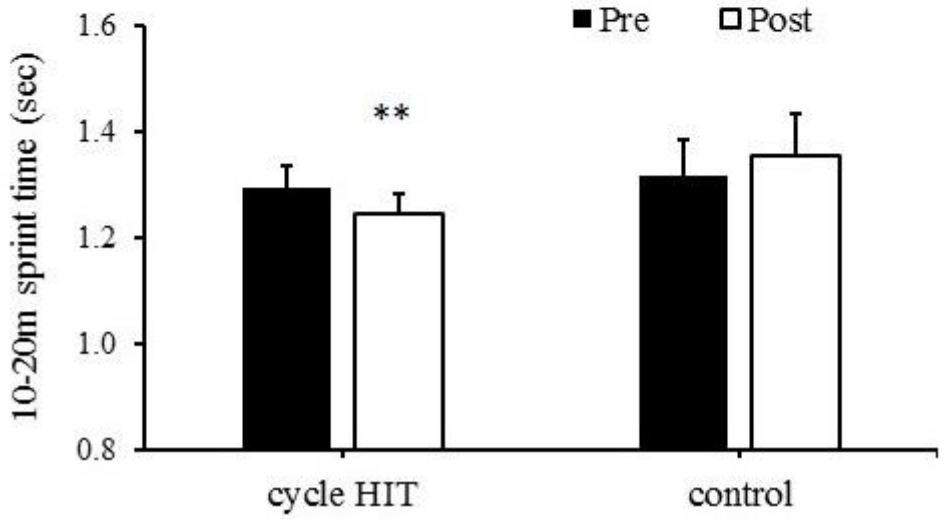
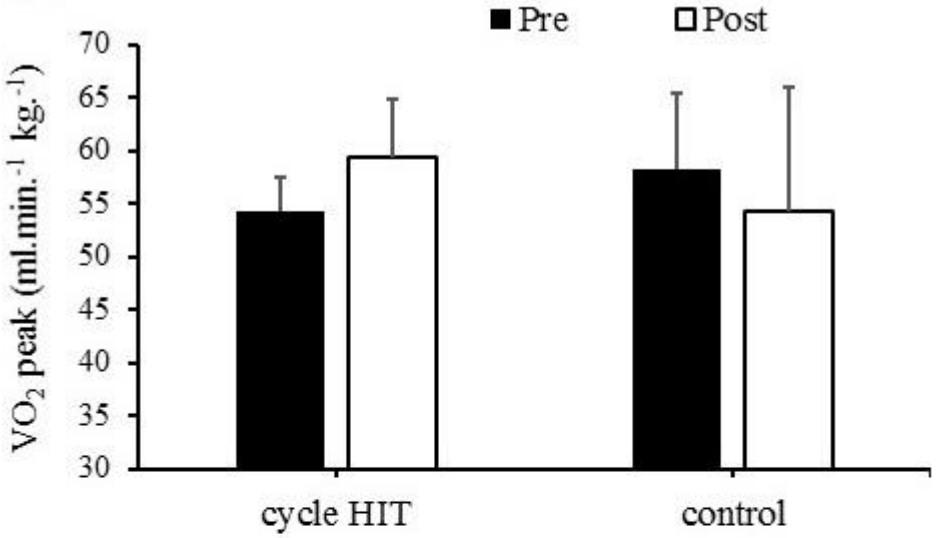


2A



2B